The Prognosis of Mitral Stenosis, Pure or Complicated, up to Fifty Years and over.

Dr. DUROZIEZ, in an article on this subject, divides his cases of diseases or lesions of the heart into two classes, those in patients up to fifty years of age, and those of fifty years and over; of the latter class, he gives 193 cases. Of these 193 cases there are 35 of pure or complicated mitral stenosis. It may be interesting to know if many who suffer from mitral stenosis pass the age of fifty years, and what effect other cardiac lesions have on cases of mitral stenosis. Can we give these patients the hope of a relative longevity? This matter has received but little attention from authors. Autopsies of persons suffering from mitral stenosis, who have attained the age of fifty years, are very rare; and the observations of non-fatal cases are not more common, although we can to-day diagnosticate mitral stenosis with about the same certainty as we can pneumonia.

Persons suffering from mitral stenosis live longer than is generally supposed. In the text-books the degree of asystolism is regarded almost to the exclusion of the personal appearances. Without doubt there are sufferings of greater or less intensity, but the patient lives. We say that the period preceding asystolism is long or very long, but this is indefinite. We say that the subject of mitral stenosis lived long, because the lesion commenced late. It is far from easy to fix upon the exact date of the commencement of a cardiac lesion; it is not sufficient to date from a certain attack of acute rheumatism, as Duroziez has had cases of mitral stenosis under observation for more than fifteen years which are still in good general health.

Classifying his 35 cases by their complications and aggregate ages we find that: 10 cases of mitral stenosis with mitral insufficiency aggregate 554 years; 9 cases of uncomplicated mitral stenosis give 487 years; 4 cases of mitral stenosis with aortic insufficiency give 232 years; 4 cases of mitral stenosis with mitral and aortic insufficiency and aortic stenosis give 225 years; 4 cases of mitral stenosis and insufficiency with aortic stenosis and insufficiency give 213 years; 1 case of mitral stenosis with aortic insufficiency and tricuspid stenosis and insufficiency gives 58 years; 1 case of mitral stenosis with mitral and tricuspid insufficiency gives 57 years; 1 case of mitral stenosis with tricuspid stenosis and mitral, aortic and tricuspid insufficiency gives 55 years; 1 case of mitral stenosis with aortic stenosis and insufficiency gives 52 years.

It is seen that mitral stenosis with mitral insufficiency heads the list with 554 years for 10 cases, with pure mitral stenosis next, 487 years for 9 cases. We find, however, that a tricuspid lesion is a very important factor in determining a fatal issue, only three living cases being given. We are also struck by the weight which aortic lesions seem to have. Lesions of the mitral valve permit the attainment of 60 years of age; those of the aortic orifice at most 50 years; those of the tricuspid scarcely 40 years. Of the 35 cases, 18 are males, and 17 females. Duroziez draws the following conclusions: 1. Mitral stenosis (so large as to permit the passage of a finger and giving rise to characteristic bruits), pure or complicated, by other valvular lesions, exceptionally permits one to pass the age of 60 years. 2. A complicating mitral insufficiency does not aggravate the prognosis. 3. A complicating stenosis does aggravate it. 4. A complicating tricuspid lesion makes the prognosis very grave.—*L' Union Méd.*, August 5, 1883.

Paroxysmal Hæmoglobinuria.

Dr. STEPHEN MACKENZIE read a paper on this subject at a late meeting of the Medical Society of London. He commenced by relating three cases. Case

1 was a boy, aged thirteen, who had always lived in the East-end of London. His disease commenced at Christmas, 1881, and lasted till August, 1882. The attacks occurred whenever he went out into the cold air, and only under these circumstances. This occasioned a chill, followed by the passing of water like porter or port wine. In a few hours the urine became quite natural. Case 2 was a man, aged fifty-five, who had an attack eighteen years before, lasting a few weeks. He remained free until the present year, when he again became affected, and the attack lasted three months. Each paroxysm was excited by exposure to cold, when the urine became porter-coloured, and in a few hours, after the patient became warm, it resumed its natural appearance. Case 3 was that of a gentleman, fifty years of age, who had suffered from the disease for twenty-three years. Each attack was invariably excited by cold. The urine between the attacks was natural in colour. This patient had had primary syphilis, but no sequelæ; the others had not; and none of the three had suffered from malaria. Dr. Mackenzie then described in detail the peculiarities of the paroxysmal and inter-paroxysmal urine in this disease. The blood colour of the urine was due to hæmoglobin, as shown by the guaiacum and Heller's tests, and spectroscopic examination, methæmoglobin being sometimes, but not always, present. A few blood-corpuscles were sometimes found, but more often they were entirely absent. The other phenomena of the paroxysm were then described, and what was known of the pathology of the disease was next reviewed. Two things were certain: (1) that blood destruction or dissolution took place somewhere; (2) that this was, in the great majority of cases, due to the influence of cold acting on a morbidly sensitive reflex nervous mechanism. Probably a second factor was present in a peculiarity of the blood-corpuscles. As to where the blood destruction took place the evidence was not quite conclusive, but some recent observations of Boas and others suggested that it was in the exposed parts affected by the cold. Allusion was made to the connection between this disease and the "local asphyxia," or symmetrical gangrene, described by Raynaud, and examples of which had been recorded by Wilks, Southey, Barlow, Mahomed, etc. It appeared possible that in some cases there was an imperfect formation of the blood-corpuscles, which, under the influence of cold, and possibly carbonic acid, parted with their hæmoglobin more readily than in health. Hæmoglobin had been shown in some cases to be present in the serum. It might be that the kidneys were passive, as whenever dissolved hæmoglobin circulated in the blood, the kidneys separated and eliminated it. This was shown in toxic conditions, as in poisoning with arseniuretted hydrogen, naphthol, chlorate of potash, etc., and in certain fevers, as well as in the hæmoglobinuria of cattle and horses. Some sections were shown of the kidney prepared by Dr. Dreschfeld, of Manchester, from a fatal case of hæmoglobinuria from chlorate of potash poisoning. It might be that in some cases the blood destruction occurred in the kidney, but it was certain in some cases that it occurred elsewhere. The few post-mortem examinations that had been made threw no light on the nature of the disease, which occurred at all ages, with a marked predominance in young males. Syphilis had been shown to have been an antecedent in several cases, and the disease had been cured in some such by antisyphilitic treatment. In others malaria was apparently the cause of the disease, though not in all. Rheumatism had been associated with the disease. It was possible in these dyscrasiæ that an imperfect blood formation took place. As regards treatment, syphilis should be inquired for, and when present treated. In the majority of cases quinine was the remedy of greatest service, even in cases evidently not malarial. Probably some patients who got well when taking quinine would have done so without it. Most patients found by experience that the only way to prevent attacks was to avoid exposure

to cold by clothing and habits. But it was possible, as suggested by Dr. Barlow, that gradual accustoming to cold might be at once the most philosophical and successful mode of treatment, and this was decidedly worthy of trial.

Dr. DICKINSON said that he had had large experience of such cases. Sixteen cases had been under his own care, and for one more he was indebted to Sir William Jenner. One fact which came out very prominently from this collection of cases was the remarkable influence which ague or malarial disease seemed to play in the causation. Five of the cases had had ague, and three had resided in a malarial district. In two, other members of the family had suffered from ague; and except in three cases, a strong presumption of malarial influence existed. It must be remembered that malarial conditions of body are very tenacious and come out whenever they have a chance, thereby earning the title of an insidious disease. Paroxysmal hæmatinuria resembled an attack of ague in many ways. It might be said metaphorically that the disease eliminated itself by the kidney rather than through the skin, as in ordinary ague. Then the association of ague and hæmatinuria had been noted, as in a case recorded by Dr. Mackenzie. Dr. Dickinson had had the case of a young woman who had contracted malaria in India under his care. There were undoubted attacks of ague associated with hæmaglobinuria, hemorrhages in various parts of the body, and purpura. Under the influence of quinine the patient got nearly well; but died some time later from perforation of a typhoid ulcer. At the autopsy, the kidneys showed signs of inflammation in the overgrowth of fibroid tissue and in the blocking of tubes. Besides there were many areas of interstitial hemorrhages to be seen around, but not in the Malpighian bodies. That cases get well of their own accord there could be no doubt. And of the value of large doses of quinine continued for a long time, Dr. Dickinson had no doubt.

Dr. Mackenzie replied that he did not ignore ague as an element in the causation of the disease. But there were cases, as in one of his own, where no suspicion of malaria could be entertained.—*Lancet*, Nov. 17, 1883.

Cases of Thickened Epidermis treated by Salicylic Plaster.

At a late meeting of the Clinical Society of London (*British Medical Journal*, December 1, 1883), Dr. THIN gave an account of cases of thickened epidermis treated by salicylic gutta-percha plaster. The plaster which he used was manufactured by Herr Beiersdorf, of Hamburg, at the suggestion of Dr. Unna, who had introduced it into practice.

The first case which he related was that of an adult man, in whom a tendency to extreme tylosis of the soles and palms was hereditary. The palms and soles in this man's case were covered with an extremely thick and hard epidermis, and had been for many years in this condition, the affection having resisted very various methods of treatment. The treatment by the plaster was, in the first instance, recommended by Dr. Unna, and the author simply continued Dr. Unna's treatment. Under the use of the salicylic plaster, which was kept constantly applied by means of bandages, and changed every third or fourth day, the hard layer of epidermis came off in one mass, leaving a delicate rose-coloured epidermis behind it. There was neither pain nor inconvenience connected with the use of the plaster, and the patient, a business-man actively employed, was able to follow his usual avocation without interruption.

CASE II.—A gentleman, aged 72, who had always been healthy, was unable to walk for a period of six or seven months on account of an attack of sciatica. When the sciatica was relieved, he somewhat suddenly resumed his professional employment in the city, which at the time involved a good deal of walking on